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Alexander Ypsilante (1792-1832)

We read in the neurological stamp column of the Journal the comments of Dr Haas1 regarding the disease of Alexander Ypsilante (1792-1832). As the writer noted the picture on the stamp in honour of Prince Alexander Ypsilante, issued by the Greek state in 1930, fails to show that he probably had myotonic dystrophy, a disease which was diagnosed in 1932 in London by Professor Caughey in a female descendant of Alexander Ypsilante²³; another four siblings from her family of eight also had the illness.

Recent in depth research on the medical history of the family of Alexander Ypsilante, ongoing in the Department of Neurology, University of Patras4-6 support Caughey's hypothesis, according to which Alexander Ypsilante, the leader of the Greek revolution-and probably some of his brothershad myotonic dystrophy. Our studies, based on correspondence, state archives, and medical documents found in Greece, Rumania, and Austria have shown that Alexander Ypsilante, his younger brother Demetrious, also a hero of the Greek struggle for independence against the Turks in the last century, and two more brothers of the remaining five children of the family, often manifested symptoms highly suggestive of dystrophia myotonica; these included early frontal baldness, severe weakness and atrophy of face and limb muscles, hypofertility, pulmonary infections, heart problems, and vision failure. Despite these data and the fact that necropsy was carried out on Alexander Ypsilante, Ypsilante brothers' disease still remains an enigma because the disease, unknown during their time, was only described later (1909), by Steinert. Hopefully with the help of modern molecular genetic techniques (DNA technology)7 and bones from the tomb of Demetrious Ypsilante, located in Nafplion, the first post-revolutionary capital of Greece, it would be possible to precisely state the nature of the Ypsilante family illness.
TH PAPAPETROPOULOS

Department of Neurology, Medical School of Patras University, el School of Failus Chicolom, PO Box 1045, 26 500 Rion, Greece

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Angiotropic lymphoma in the differential diagnosis of systemic vasculitis

We were interested to read the report by Roux et al of angiotropic lymphoma with mononeuritis multiplex mimicking systemic vasculitis.1 We have recently published a case with striking similarities to this one.2 Two other cases not mentioned by the authors have also mimicked vasculitis.34 In our patient even the antineutrophil cytoplasmic antibody titre test was repeatedly positive emphasising the point made by Roux et al that this condition may mimic a systemic necrotising vasculitis extremely closely. In both cases the patient had a rash suggestive of livedo reticularis, a progressive flaccid paraplegia, loss of sensation in the legs, urinary retention, and anal incontinence. Both patients also developed panhypopituitarism, massive oedema, hypotension with profound hyponatraemia. Antineutrophil cytoplasmic antibody titres are found in several inflammatory conditions and are often present in systemic vasculitis.5 In our case, the combination of such titres and clinical features pointed us incorrectly towards this diagnosis. We agree strongly with the authors that the differential diagnosis of systemic necrotising vasculitides must include angiotropic lymphoma, particularly as this condition is difficult to diagnose before death, had a dismal prognosis if undiagnosed, and is potentially curable.

AMMAR AL-CHALABI University Department of Neurology and Neuroscience, Institute of Psychiatry and Kings College School of Medicine and Dentistry, Denmark Hill, London SE5 8AF, UK

> RICHARD J ABBOTT Department of Neurology, Leicester Royal Infirmary, Leicester LE1 5WW, UK.

Correspondence to: Dr Al-Chalabi.

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